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# Detraining Produces Minimal Changes in Physical Performance and Hormonal Variables in Recreationally Strength-Trained Men

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**ABSTRACT**

The object of this study was to examine changes in muscular strength, power, and resting hormonal concentrations during 6 weeks of detraining (DTR) in recreationally strength-trained men. Each subject was randomly assigned to either a DTR ($n = 9$) or resistance training (RT; $n = 7$) group after being matched for strength, body size, and training experience. Muscular strength and power testing, anthropometry, and blood sampling were performed before the experimental period (T1), after 3 weeks (T2), and after the 6-week experimental period (T3). One-repetition maximum (1RM) shoulder and bench press increased in RT at T3 ($p \leq 0.05$), whereas no significant changes were observed in DTR. Peak power output and mean power output significantly decreased (9 and 10%) in DTR at T2. Peak torque of the elbow flexors at 90° did not change in the RT group but did significantly decrease by 11.9% at T3 compared with T1 in the DTR group. Vertical jump height increased in RT at T2 but did not change in DTR. Neither group displayed any changes in 1RM squat, body mass, percent body fat, or resting concentrations of growth hormone, follicle-stimulating hormone, luteinizing hormone, sex hormone–binding globulin, testosterone, cortisol, or adrenocorticotropin. These data demonstrate that 6 weeks of resistance DTR in recreationally trained men affects power more than it does strength without any accompanying changes in resting hormonal concentrations. For the recreational weight trainer, losses in strength over 6 weeks are less of a concern compared with anaerobic power and upper arm isometric force production. Anaerobic power exercise with a high metabolic component coming from glycolysis might be of importance for reducing the impact of DTR on Wingate power performances. A minimal maintenance training program is recommended for the recreational lifter to offset any reductions in performance.

***Key Words:*** anaerobic, Wingate test, 1 repetition maximum, power, endocrine

***Reference Data:*** Kraemer, W.J., L.P. Koziris, N.A. Ratamess, K. Häkkinen, N.T. Triplett-McBride, A.C. Fry, S.E. Gordon, J.S. Volek, D.N. French, M.R. Rubin, A.L. Gómez, M.J. Sharman, J.M. Lynch, M. Izquierdo, R.U. Newton, and S.J. Fleck. Detraining produces minimal changes in physical performance and hormonal variables in recreationally strength-trained men. *J. Strength Cond. Res. 16(3):373–382. 2002.*

## Introduction

Cessation of resistance training (RT) or significant reduction of training volume, intensity, or frequency results in detraining (DTR) (4). Physiological changes corresponding to decreased performance capacity have been reported after periods of significantly reduced training (13), complete cessation of training (2, 37, 38), and immobilization (26, 27). The magnitude may be dependent upon the length of the DTR period (6, 7, 9) and how highly trained the individual is (15, 35). Considering that periods of DTR are common among athletes but may be more so for individuals who exercise regularly for fitness purposes, under-

standing the effects of training cessation or DTR is as important in training for muscular fitness as well.

It appears that DTR is a complex physiological state, which may entail several mechanisms contributing to performance decrements. Previous investigations have shown significant decreases in muscular strength (1, 15, 35) after DTR periods of 2–32 weeks but not to the extent of pretraining values (14, 16), indicating a slower ''DTR effect'' than the rate at which initial improvements occur. In addition, strength retention is greater when eccentric muscle actions are included (3, 14, 16). The mechanisms of strength loss appear less clear. Decreases in maximal integrated electromyography (IEMG) have been reported between 2 and 12 weeks of DTR (6, 7, 9, 15). It has been proposed that the mechanisms involved with the DTR-related strength reductions may involve predominantly neural changes initially with a gradually increasing role of atrophy during long-term periods of DTR (7).

Decreases in muscle fiber size (9, 15, 21, 36) with concomitant changes in fiber-type composition (35, 36) have been reported after periods of DTR. Häkkinen et al. (9) and Staron et al. (35) reported significant decreases in both slow- and fast-twitch muscle fiber area after DTR in recreationally trained lifters and a highly trained power lifter. Hortobagyi et al. (15) reported a significant decrease in fast-twitch muscle fiber size after only 2 weeks of DTR in highly trained power lifters and football players. Thus, atrophy is a contributing factor to DTR-related strength loss and may be more apparent in individuals who have experienced a significant magnitude of hypertrophy.

Few studies have examined changes in hormonal concentrations after DTR. No significant differences in testosterone (T), cortisol (C), sex hormone–binding globulin, luteinizing hormone, growth hormone (GH), follicle-stimulating hormone, and thyroid hormones have been reported during 8–12 weeks of DTR (11, 13, 30). In addition, Häkkinen et al. (11) reported a significant reduction in the T/C ratio in men, which correlated highly with strength decreases. In contrast, Hortobagyi et al. (15) reported significant increases in GH, T, and the T/C ratio with a significant decrease in C after 2 weeks of DTR in highly trained power lifters and football players. They hypothesized that this increase in anabolic hormones was related to the body's ability to combat the catabolic processes associated with DTR. However, it is possible that the increase in circulating hormones may not result in anabolism at the tissue level (24). Thus, the hormonal response to DTR appears to be varied and may depend upon how highly the individual is trained and the individual's recent training history (4).

Little is known concerning the effects of DTR on muscle power determined using the Wingate test. Considering that force is an integral component of power and most studies report significant reductions in force production with DTR (6, 9, 29), it seems reasonable to hypothesize that power may be reduced with DTR, especially when it is associated with high lactate concentrations (e.g., Wingate test). Research investigating changes in vertical jump performance after DTR have shown no changes after 2 weeks (15) and a 3–5% reduction after 12 weeks of DTR (1). In addition, Häkkinen et al. (6, 9) reported no or minor changes in rate of force development after 8 and 12 weeks of DTR, and Ishida et al. (18) reported an increase in rate of force development after 8 weeks of DTR. It appears that fast force–producing capacity may be unaltered or enhanced with short-term DTR periods, but peak anaerobic power decrements in the Wingate test are less clear.

Previous studies have only partially addressed the effects of DTR in individuals who are primarily training to enhance recreational muscular fitness and health. Thus, the object of this investigation was to examine the impact of 6 weeks of DTR on muscle strength and power performances in men involved with recreational RT. A secondary purpose was to examine resting serum hormonal concentrations to determine if any evidence might implicate an underlying hormonal basis for DTR effects.

## Methods

### *Experimental Approach to the Problem*

In order to address the primary hypothesis presented in this investigation, we selected men who were involved with RT for improving health and muscular fitness. Each subject had been training consistently over the past year and was matched according to training history (i.e., years of experience), body size, and muscular strength. Subsequently, each participant was randomly assigned to either a DTR group ($n = 9$) or a RT group ($n = 7$). The DTR group discontinued RT and did not perform any resistance or sprint exercise (including manual labor) or formal exercise throughout the 6-week experimental period. The RT group continued its regular resistance training workouts during the 6-week period. Thus, this study design enabled us to make comparisons between 2 similar groups of recreationally resistance-trained men, 1 that continued training and 1 that completely stopped training for 6 weeks. Resistance training leading up to the detraining period was performed 2–4 days per week and consisted of exercises stressing all major muscle groups using multiple sets (i.e., 3–5 per exercise) for 6–10 repetitions with loads corresponding to 70–85% of 1 repetition maximum (1RM). Assessments for anthropometry, muscular strength, power, and resting hormonal concentrations were performed immediately before the experimental DTR period, after 3 weeks, and at the conclusion of the 6-week DTR period.

**Table 1.** Subject characteristics.*

| | RT ($n$ = 7) | DTR ($n$ = 9) |
|---|---|---|
| Age (y) | 21.9 ± 1.9 | 21.1 ± 0.6 |
| Height (cm) | 178.4 ± 2.0 | 175.6 ± 2.2 |
| Body mass (kg) | 76.3 ± 7.9 | 79.4 ± 3.7 |
| Body fat (%) | 13.0 ± 3.1 | 17.5 ± 1.8 |

* RT = resistance training; DTR = detraining.

### *Subjects*

The subjects consisted of healthy men between the ages of 18 and 35 years. The risks of the study were explained to the subjects before participation, and each subject signed an institutionally approved informed consent document before participation in the investigation. The physical characteristics of the subjects are presented in Table 1. Two subjects in the RT group dropped out because of schedule conflicts. Each subject was involved in recreational (noncompetitive) RT 2–4 days per week for a minimum of 2 years. Furthermore, training was monitored during the preceding 6 weeks to ensure a consistent baseline of training leading into the DTR period so that no subject initiated the study in a detrained state. Subjects were familiarized with all testing procedures before the start of the testing.

### *Testing Procedures*

Assessments for body mass and composition, girth measurements, vertical jump performance, upper and lower body strength, anaerobic power, and resting hormonal concentrations were performed in the present study. No strenuous exercise was permitted in the 48-hour period before each testing session, and no alcohol consumption was allowed during the final 24 hours. All subjects were tested at the identical time of day for each testing time point in the study. All testing sessions lasted approximately 2.5 hours and were conducted between 0900 and 1430 hours. The rationale for the order of testing was based on the level of fatigue associated with each test, and we started with tests of lowest fatigue. Test-retest reliability for the order we used demonstrated high intraclass Rs ($R \geq 0.93$). In addition, each subject was accustomed to performing maximal attempts. Therefore, all subjects underwent the testing protocol in the same order described as follows: (a) Blood draw (0900–1200 hours), (b) Anthropometry: height, body mass, percent body fat, and girth measurements, (c) Vertical jump (30 minutes rest), (d) Isokinetic and isometric force (30 minutes rest), (e) 1RM bench press (60 minutes rest), (f) 1RM squat (60 minutes rest), (g) 1RM behind-the-neck shoulder press (60 minutes rest), (h) Wingate anaerobic power cycle ergometer.

### *Anthropometry*

Body density was estimated with a Lange skinfold caliper (Country Technology, Gays Mills, WI) using the 7-site method previously described (19). The sites measured were the pectoral, triceps, midaxillary, subscapular, abdominal, suprailiac, and thigh. Percent body fat was estimated using the equation of Siri (34). Girth measurements were obtained for the chest, hip, proximal thigh, upper arm, and waist using a Gulick anthropometric tape measure according to methods previously described (31). The same investigator performed all skinfold and girth measurement assessments.

### *Vertical Jump Performance*

Counter-movement vertical jump and reach height (CMVJ) was measured using a Vertec (Sports Imports, Inc., Columbus, OH). Each subject stood flat-footed and extended his arm as high as possible. This starting height was recorded, and the distance was calculated. Each subject then proceeded to perform a maximal CMVJ, in the process touching the highest marker on the Vertec as possible. This area was marked, and the distance was calculated. A 2-ft takeoff was used, and no approach steps were permitted. Maximal CMVJ height was determined by subtracting the starting height from the height attained during the jump. After 3 practice jumps, 3 trials were performed, and the highest score was recorded.

### *Muscular Strength*

The procedures used for isometric maximal strength testing have been outlined by Sale (32). Isometric strength of the elbow and knee extensors and flexors was assessed using a Cybex II dynamometer (Lumex Corp., Ronkonkoma, NY). Peak isometric torque was measured at a knee angle of 45° and at an elbow angle of 90°. Subjects were instructed to exert maximal force for 5 seconds for 3 trials, with the highest torque recorded for further analysis.

Upper and lower body dynamic muscular strength was measured using the 1RM bench press, behind-the-neck press, and squat (23). Briefly, each subject performed 2 warm-up sets with 40–60% and 60–80% of his perceived 1RM for each exercise. Each subsequent set was performed for 1 repetition as the load was progressively increased until the subjects reached their respective 1RM. All 1RMs were determined within 5 sets to avoid excessive fatigue. The exercise order was the bench press, squat, and behind-the-neck press. Standardized techniques and procedures (i.e., grip-stance widths, range of motion, body position, and biomechanics) were used for all 3 exercises. Any lifts failing to meet standardized criteria were discarded.

### *Wingate Anaerobic Power Test*

The Wingate anaerobic power test was performed on a cycle ergometer (Monark Ergometer Model 818E;

Monark AB, Varberg, Sweden) anchored to the floor and modified to permit the instantaneous application of an opposing force of 0.736 $N \cdot kg^{-1}$ body mass. Seat height was adjusted such that the knees were slightly bent (approximately 10°) when the pedal of the same side was at its lowest position during a revolution. Subjects performed a 2-minute warm-up using a self-selected resistance and cadence followed by 1 minute of rest. Subsequently, subjects were instructed to pedal as rapidly as possible with maximal effort against the inertial resistance of the flywheel, with resistance being applied to initiate the test. Subjects were required to remain seated throughout the 30-second bout, and verbal encouragement was provided during the test. Flywheel revolutions were monitored and recorded for each of six 5-second segments via an electromagnetic detection system with printer interface (Miniprinter Model #MM2481/5S1; Keltron Corporation, Waltham, MA). Power output was calculated using the number of flywheel revolutions and the opposing force. Peak power was determined as the highest 5-second segment average power output. Mean power was computed using the total number of revolutions for the 30-second bout. Data for Wingate tests were obtained only at T1 and T2 because of a technical problem at T3.

### *Blood Sampling*

Venous blood samples were obtained from subjects kept in a semirecumbent position upon arrival for the testing session after an overnight fast, at T1, T2, and T3. Venous blood samples were obtained from a superficial arm vein on the radial aspect of the arm using a 20-gauge needle and syringe vacutainer setup. Before obtaining a resting blood sample, a 20-minute equilibration period was allowed to lapse. Blood samples were obtained at the same time of day for each subject (between 0900 and 1200 hours) to reduce any possible effects of diurnal variations on hormonal concentrations. All blood samples were centrifuged at 1,500*g* for 15 minutes, with serum and plasma samples harvested, and stored at −80° C until analysis.

### *Biochemical Analyses*

Resting serum concentrations of C, T, and sex hormone–binding globulin (SHBG) were determined via a single-antibody solid phase $^{125}I$ radioimmunoassay (Diagnostic Products Corp., Los Angeles, CA) with detection limits of 5.3, 0.38, and 6.0 $nmol \cdot L^{-1}$, respectively. An LKB Model 1272 Clini gamma counter and on-line data reduction system (Pharmacia LKB Nuclear, Inc., Gaithersburg, MD) was used to determine immunoreactivity values. Resting serum follicle-stimulating hormone (FSH), leutenizing hormone (LH), and plasma adrenocorticotrophic hormone (ACTH) concentrations were measured using a liquid phase radioimmunoassay with double-antibody technique (Diagnostic). Resting serum human GH concentrations were determined in duplicate via double-antibody $^{125}I$ radioimmunoassay (Diagnostic). All samples were assayed in duplicate and were decoded only after analyses were completed (i.e., blinded analysis procedure). Intra-assay variance was less than 5% for all these hormones. All samples for any one hormone were analyzed within the same assay to eliminate the effect of interassay variance. In addition, samples were only thawed once before analysis.

### *Statistical Analyses*

A two-way (group × time) analysis of variance with repeated measures was used to analyze changes in all variables between time points. When appropriate, Tukey's post hoc analyses were used to determine pairwise differences between T1, T2, and T3. Using n-Query Advisor® software (Statistical Solutions, Saugus, MA) the statistical power values calculated for each dependent variable at the $n$ sizes used ranged from 0.79 to 0.85 at the 0.05 alpha level. Pearson product-moment correlational analyses were used to examine various bivariate relationships. The statistical significance condition chosen in this study was $p \leq 0.05$. Data are reported as the means ± 1*SD*.

## Results

### *Anthropometry*

Body mass did not change significantly in either group (RT = 76.3 ± 21.0 pre, 76.8 ± 20.7 post; DTR = 79.4 ± 11.2 pre, 79.0 ± 10.7 post). Percent body fat increased in both groups but none of these increases were significant (RT = 13.0 ± 8.2% pre, 13.8 ± 7.5% post; DTR = 17.5 ± 5.5% pre, 18.3 ± 5.5% post). No significant differences were observed for hip, waist, arm, thigh, and chest circumferences in either group.

### *Dynamic Muscular Strength*

The results for changes in dynamic muscular strength throughout the 6-week experimental period are presented in Figure 1 (panels A, B, and C). No significant differences were observed for the bench press and behind-the-neck press 1RM at T2 in either group. However, significant increases in bench press and behind-the-neck press 1RMs were observed for the RT group at T3. The decreases observed for both these exercises in the DTR group were not significant. No significant differences in squat 1RM were observed in either group. When the 1RMs for all 3 exercises were totaled, the RT group values increased significantly from T1 to T3 (223.4 ± 35.7 to 237.7 ± 43.5 kg) whereas the DTR group values showed an insignificant ($p = 0.14$) decrease (231.5 ± 31.6 to 226.1 ± 27.3 kg). When the ratio of the total load lifted to body mass was calculated, a trend ($p = 0.06$) to increase was observed in the RT group (3.06 ± 0.8 to 3.19 ± 0.7), whereas an insignif-

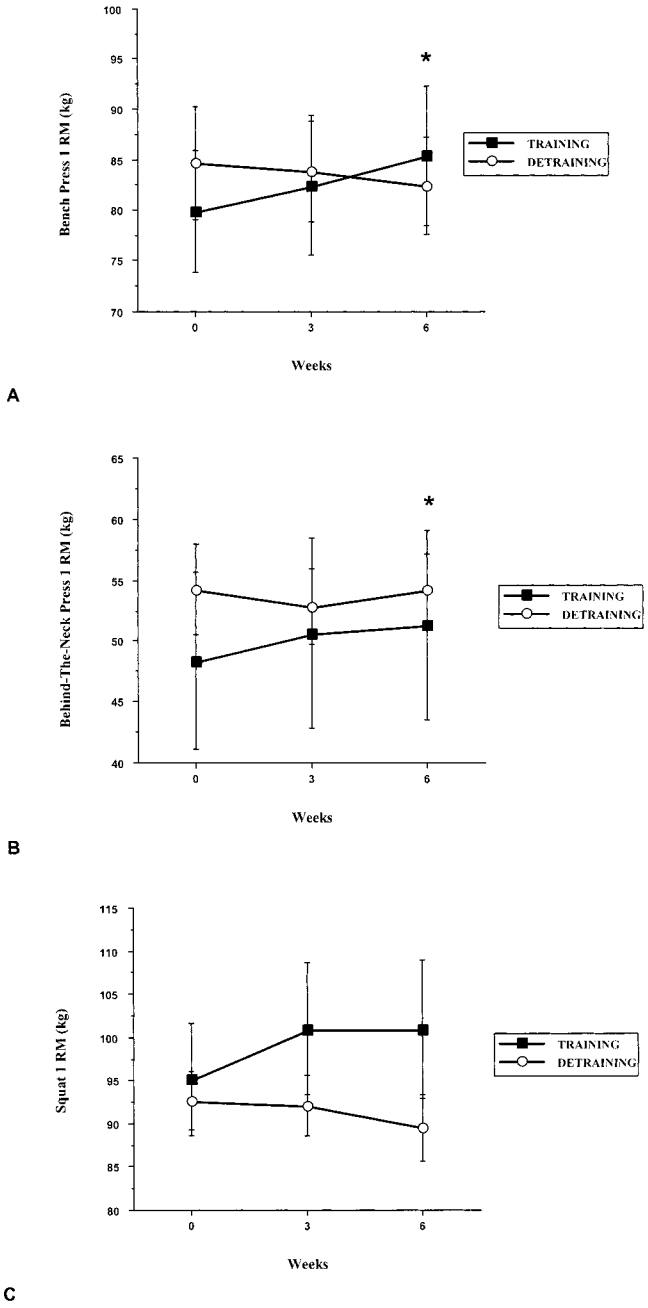


**Figure 1.** Changes in 1RM strength during the 6-week experimental period. Panel A depicts changes in 1RM bench press. Panel B depicts changes in 1RM behind-the-neck press. Panel C depicts changes in 1RM squat. *$p <$ 0.05 from corresponding time point T1 for the training (RT) group.

icant decrease was observed for the DTR group (2.98 ± 0.4 to 2.94 ± 0.4).

### *Isometric Peak Torque*

Data for isometric peak torque of the elbow and knee extensors and flexors are presented in Table 2. Peak torque of the elbow extensors at 90° did not change in the RT group but decreased by 17.5% in the DTR group. However, only a trend was observed for this decrease ($p = 0.10$) between T3 and T1. Peak torque of the elbow flexors at 90° did not change in the RT group but did significantly decrease by 11.9% at T3 compared with T1 in the DTR group. Peak torque of the knee extensors at 45° decreased insignificantly in both groups between T3 and T1. Peak torque of the knee flexors did not change in the RT group but showed a 9.2% decrease in the DTR group between T1 and T3. This decrease was not statistically significant.

**Table 2.** Changes in isometric peak torque during training and detraining.†

| | RT (mean ± *SD*) | DTR (mean ± *SD*) |
|---|---|---|
| Elbow extension peak torque (N·m) | | |
| T1 | 76.7 ± 38.1 | 62.8 ± 9.5 |
| T2 | 68.9 ± 15.7 | 56.3 ± 15.7 |
| T3 | 71.6 ± 22.0 | 51.8 ± 13.9 |
| Elbow flexion peak torque (N·m) | | |
| T1 | 84.8 ± 17.6 | 75.5 ± 13.1 |
| T2 | 82.7 ± 14.1 | 70.0 ± 12.6 |
| T3 | 84.2 ± 12.5 | 66.5 ± 14.8* |
| Knee extension peak torque (N·m) | | |
| T1 | 221.0 ± 23.2 | 190.6 ± 51.8 |
| T2 | 204.2 ± 47.9 | 196.7 ± 39.6 |
| T3 | 194.6 ± 20.1 | 183.8 ± 41.5 |
| Knee flexion peak torque (N·m) | | |
| T1 | 158.4 ± 24.1 | 133.3 ± 38.6 |
| T2 | 162.2 ± 20.6 | 127.9 ± 40.0 |
| T3 | 157.7 ± 26.8 | 121.1 ± 31.2 |

† RT = resistance training; DTR = detraining.
* $p < 0.05$ from corresponding time point T1.

**Table 3.** Changes in anaerobic power and vertical jump during training and detraining.†

| | RT (mean ± *SD*) | DTR (mean ± *SD*) |
|---|---|---|
| Peak power (W) | | |
| T1 | 802.4 ± 128.8 | 859.0 ± 111.2 |
| T2 | 802.7 ± 186.2 | 784.3 ± 143.2* |
| Mean power (W) | | |
| T1 | 468.3 ± 67.4 | 503.9 ± 79.9 |
| T2 | 468.3 ± 54.6 | 452.4 ± 99.8* |
| Vertical jump height (cm) | | |
| T1 | 46.6 ± 9.7 | 44.6 ± 5.8 |
| T2 | 50.4 ± 8.2* | 46.8 ± 7.0 |
| T3 | 49.7 ± 10.3 | 46.7 ± 8.5 |

† RT = resistance training; DTR = detraining.
* $p < 0.05$ from corresponding time point T1.

### *Anaerobic Power and Vertical Jump Performance*

Changes in anaerobic power and vertical jump performance are presented in Table 3. Both peak power and

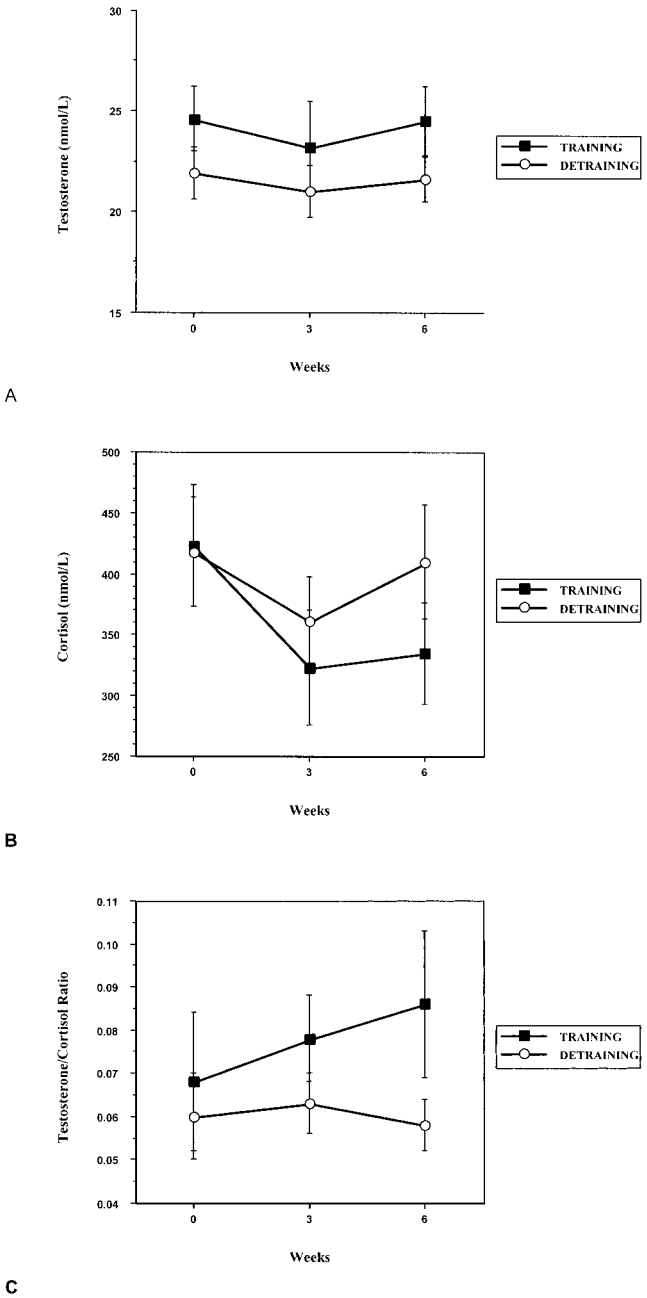


**Figure 2.** Changes in resting serum hormonal concentrations during the experimental period. Panel A depicts the response of T. Panel B depicts the response of C. Panel C depicts the T/C ratio. No differences were observed for any of these hormones throughout the experimental period.

mean power obtained via the Wingate test did not change in the RT group between T1 and T2. However, significant decreases were observed in the DTR group between T1 and T2 for both peak power and mean power (8.7 and 10.2% respectively). It is important to note that the Wingate test was not performed at T3. Vertical jump height significantly increased in the RT group at T2 but did not show any further change at T3. No significant difference was observed in the DTR group.

**Table 4.** Resting hormone concentrations over the experimental period (mean ± *SD*).*

| Group | T1 | T2 | T3 |
|---|---|---|---|
| ACTH (pmol·L$^{-1}$) | | | |
| CRT | 4.2 ± 5.0 | 2.4 ± 3.1 | 4.3 ± 5.0 |
| DTR | 4.9 ± 3.0 | 3.8 ± 4.0 | 2.7 ± 3.0 |
| GH (μg·L$^{-1}$) | | | |
| CRT | 1.4 ± 2.4 | 1.0 ± 1.7 | 0.8 ± 0.9 |
| DTR | 2.0 ± 2.0 | 1.35 ± 1.0 | 0.6 ± 1.1 |
| FSH (IU·L$^{-1}$) | | | |
| CRT | 6.2 ± 4.0 | 5.8 ± 3.6 | 6.4 ± 4.5 |
| DTR | 6.2 ± 3.3 | 6.5 ± 4.1 | 6.1 ± 4.5 |
| LH (IU·L$^{-1}$) | | | |
| CRT | 3.2 ± 1.5 | 5.0 ± 2.7 | 5.2 ± 2.2 |
| DTR | 3.9 ± 1.9 | 4.3 ± 1.9 | 3.7 ± 2.1 |
| SHBG (nmol·L$^{-1}$) | | | |
| CRT | 21.2 ± 17.0 | 15.1 ± 11.7 | 21.5 ± 17.8 |
| DTR | 15.5 ± 12 | 17.2 ± 12.0 | 15.1 ± 11.4 |

* ACTH = adrenocorticotropin; CRT = Continued Resistance Training group; DTR = Detraining group; GH = growth hormone; FSH = follicle-stimulating hormone; LH = luteinizing hormone; SHBG = sex hormone binding globulin.

### *Resting Hormone Concentrations*

Changes in T, C, and the T/C ratio are presented in Figure 2 (panels A, B, and C). No significant differences in resting serum T concentrations were observed for either group. Resting serum C concentrations did not significantly change in the DTR group but did decrease by 20.8% in the RT group, whereas the DTR group showed no significant decrease. Likewise, there was an insignificant increase in the T/C ratio for the RT group, and no changes were observed in the DTR group. In addition, there were no significant differences observed in resting serum concentrations of SHBG, LH, FSH, and GH, and no changes were observed in resting plasma concentrations of ACTH at any time point (see Table 4).

## DISCUSSION

To our knowledge, this investigation is the first to show that 6 weeks of detraining can decrease anaerobic power (determined by the Wingate test) and peak isometric torque production of the elbow extensor and flexor muscles, although causing minimal declines in dynamic muscular strength in recreationally trained men. All of these changes occurred in the absence of any significant changes in resting serum hormone concentrations. These data show the rapidity of the loss

of isometric force and dynamic power with detraining and demonstrate the need for a maintenance program of RT to reduce the impact on power and isometric force production levels in recreational lifters.

Previously, Häkkinen and Komi (7) and Häkkinen et al. (6, 9) reported no or only minor changes in isometric fast force production during DTR periods of 8–12 weeks. In addition, Ishida et al. (18) reported a greater isometric rate of force development after 8 weeks of DTR. Considering that only minor decreases were observed for lower body force production during the first 3 weeks of DTR in the present study, it does appear that the rate of force application was reduced. Several studies have reported decreased IEMG during short-term DTR periods (6, 9). Häkkinen and Komi (7) also hypothesized that reductions in neural activity were most responsible for performance decrements early in DTR. Decreased motor unit activity may have contributed to the significant reductions observed in both peak power and mean anaerobic power in this study. In addition, MacDougall et al. (27) and Green et al. (5) reported increased resting concentrations of muscle creatine, creatine phosphate, and adenosine triphosphate (ATP) after training that were significantly reduced during subsequent 5- and 6-week periods of immobilization and DTR, respectively. It has been suggested that changes in phosphate content and splitting mechanisms may be significant for sustaining the energy turnover needed during high short-term power output activities (5). Physiologically, high-energy phosphates might also be theorized as a contributing factor to the observed decrease in anaerobic power output.

In agreement with some previous studies, recreationally trained athletes can maintain or suffer a slight decay in their neuromuscular performance during short periods of training cessation (7, 8, 15), but the training level may have influenced this lack of dramatically significant declines. In the present study, dynamic muscular strength and vertical jump performance were retained after 6 weeks of resistance DTR. Consistent with the data from the current study, Housh et al. (16) reported that strength was maintained during an 8-week DTR period in previously untrained men. Hortobagyi et al. (15) reported no significant changes in dynamic strength, isometric and isokinetic concentric knee extension force, and vertical jump in 12 power-trained athletes after only 2 weeks of training cessation. Häkkinen and Komi (8) reported a 10% decrease in 1RM squat in Olympic-style weightlifters after 4 weeks of DTR. Results from the same research group have also shown that strength athletes decreased maximal force production after 2.5 weeks of training cessation but reported minor increase in force production for normal, physically active men during the same experimental period. It has been demonstrated that previously untrained men maintained their strength performance during an 8-week detrained period (7, 8). On the basis of these data, it does appear that advanced lifters (i.e., with higher levels of training and absolute strength levels) show a greater magnitude of strength loss with detraining compared with untrained or moderately trained individuals. Without any DTR from prior plyometrics or explosive training (i.e., subjects performed no power training), our data indicate that vertical jump performance can be maintained over 6 weeks of training cessation. It is also likely that DTR effects on explosive jumping performance may occur more rapidly after explosive-type resistance or plyometric training programs (e.g., for basketball or volleyball), where vertical jump may be a more highly trained variable and therefore more susceptible to effects of training cessation.

It has been shown that reductions in neural activity (i.e., IEMG activity) were most responsible for performance decrements during the early phase of DTR (i.e., 2–3 weeks) (6–8, 15), followed by a progressive diminution of Type I and Type II muscle fiber areas and muscle mass (6, 9) during the later phases of training cessation. This may be explained in part by the greater specific tension exhibited by the well-trained weight lifters and the significant declines observed during the early phases of training cessation in comparison with recreationally trained men. Although the subjects in the present study had RT experience, they were not as strong as the subjects involved in the studies of Hortobagyi et al. (15), Häkkinen et al. (12), and Häkkinen and Komi (8). The relatively low level of 1RM strength possessed by the recreationally trained subjects in the present study may have blunted the larger theoretically expected losses observed in more advanced lifters.

Recently acquired strength gains appear to be lost at different rates depending on the type of strength performance measured. Indeed Weir et al. (38) reported more pronounced decrements in isometric strength compared to dynamic strength (1RM) following 8 weeks of DTR. Dudley et al. (3) reported a significant decrease in functional strength (i.e., 3RM leg press and leg extension) in previously untrained subjects after 4 weeks of detraining. Similar data were also reported by Houston et al. (17) in a 10-week 1-leg dynamic strength training and 12-week DTR study, showing that no significant amounts of newly acquired peak torque gains of the trained (39–60%) and untrained (12–37%) legs were lost 4 weeks after training cessation. It was also interesting to observe that peak torque output remained above pretraining levels 12 weeks after training stoppage, despite a progressive decrease in the trained (16–21%) and untrained (10–15%) leg torques. Häkkinen and Komi (7) and Häkkinen et al. (6, 9) reported that athletes could maintain or suffer only minor changes in rapid isometric force production during DTR periods of 8–12 weeks. Finally, it is worth noting that in subjects accustomed to weight

training without competitive purpose, Ishida and coworkers (18) demonstrated a slight decrease in maximal voluntary isometric contraction force and a large (22%) increase in the maximal rate of torque development 8 weeks after training cessation after 8 weeks of dynamic strength training of the calf muscle. The results of the present study support these data because isometric peak torque of the elbow extensors and flexors decreased to a greater extent (12 and 18%, respectively) than did dynamic 1RM leg strength. It could be also hypothesized that the differences in the effects of training cessation on neuromuscular performance of the upper and lower extremity muscles might be explained by the differences in the pattern or intensity of daily physical use in normal life. Thus, it is possible that the lower extremity muscles, owing to their weight-bearing role during some daily activities (i.e., walking, standing position), would be more likely exercised and therefore contributed to the strength retained than did the upper body muscles, which are used less frequently.

No differences were observed in body mass, percent body fat, or circumference measurements throughout the experiment period in either group. Hortobagyi et al. (15) reported no changes in body mass and percent fat after 2 weeks of DTR. Häkkinen and Komi (8) and Häkkinen et al. (10) reported no change in percent body fat and body mass after 8 and 12 weeks of DTR in recreational lifters. However, Colliander and Tesch (1) reported a significant increase in body fat, with no change in girth measurements, and Häkkinen et al. (6) reported minor decreases in lean body mass and thigh girth, with a slight, insignificant increase in percent body fat after 12 weeks of DTR. Thus, it appears that the initial training level of the subjects and the length of the DTR period, as well as diet, significantly affect the magnitude of change in anthropometry.

Both anabolic and catabolic hormones play important regulatory roles in skeletal muscle growth and tissue remodeling (22). Resistance training has been shown to exert potent effects on T, C, the T/C ratio, and GH conducive to increasing muscular hypertrophy (11, 24). In addition, Häkkinen et al. (11) reported a high correlation between changes in the T/C ratio and changes in muscular strength. However, the time course of hormonal alterations in DTR is less clear.

No significant differences were observed in resting serum hormones in the DTR group in the present study. These results support the findings of Häkkinen et al. (11), who reported no significant changes in T, C, SHBG, LH, FSH, or GH but did report a significant decrease in the T/C ratio after 12 weeks of DTR. In contrast, Hortobagyi et al. (15) reported significant elevations in GH, T, and the T/C ratio with a significant concomitant decrease in C after 2 weeks of DTR. These authors hypothesized that this initial increase in anabolic hormone concentrations was initiated to combat the catabolic processes of detraining. In addition, they suggested that short-term detraining might also represent an augmented stimulus for tissue remodeling and repair. Häkkinen et al. (10) reported that reduced training volume (over a 2-week period) after stressful preparatory training slightly increased the T/C ratio in elite weight lifters. We observed an insignificant increase in the T/C ratio after 3 weeks of DTR and an insignificant decrease between weeks 3 and 6. It is possible that DTR periods greater than 2–3 weeks may decrease the anabolic hormonal response. This decrease may coincide with the muscular atrophy observed during DTR periods of at least 2 weeks (9, 15, 29). However, more research on hormonal mechanisms during DTR is warranted. Considering the loss of muscle size associated with DTR, it appears plausible that the magnitude of atrophy may be partially dependent upon hormonal changes, yet these changes may not be reflected at the level of the resting circulating hormonal concentrations but rather at the level of the affected tissues (e.g., muscle) on the receptor level where cellular interactions with circulating hormones occur.

It is almost paradoxical to note that vertical jump performance and maximal 1RM squat did not change despite significant decreases in the Wingate peak power output. A plausible explanation for the phenomenon could be that other factors are related to cycling performance. Jaric et al. (20) also reported that maximal isometric force and rate of force development of the hip and knee extensors and plantar flexors only accounted for 38% of the kinematic variance observed during the vertical jump. Thus, other factors (i.e., jumping technique) may be critical for vertical jump performance and may have contributed to the lack of change despite the reduced Wingate peak power output observed in the present study. Conversely, the decreases in Wingate mean power may be due to a concomitant DTR of the acid-base buffering system, whereby power output is reduced within the context of an acidic environment. This finding has significant relevance to athletes involved in anaerobic sports, specifically during recovery from a sport-specific injury and during periods between 2 training seasons. To our knowledge, this is the first investigation to show significant reductions in anaerobic power (determined by the Wingate test) during short-term strength training cessation.

Training cessation has also been reported to induce small nonsystematic changes in the maximal anaerobic power in highly trained and recently trained (recreationally) and sedentary individuals. Simoneau et al. (33) observed that 7 weeks of DTR provoked significant decreases in maximal 90-second ergocycle performance, whereas the interruption of training had no effect on glycolytic enzyme markers (PFK and LDH)

after a 15-week training program. In contrast, Linossier et al. (25) reported no significant DTR-induced decreases of newly acquired maximal short-term power output and glycolytic potential (i.e., phosphorylase, phosphofructokinase, and lactate dehydrogenase activities) within 7 weeks of training cessation after 9 weeks of cycle short-sprint training. Houston et al. (17) did not observe any significant changes in the activities of enzymes representative of phosphagen (creatine kinase) and glycolytic (hexokinase, phosphofructokinase, and lactate dehydrogenase) metabolism, neither after 10 weeks of dynamic strength training nor after 12 consecutive weeks of training cessation. It has been reported that mitochondrial enzyme and glycogen synthase activities decline to pretraining levels after short-term training-DTR protocols (21, 28). In addition, MacDougall et al. (26) and Green et al. (5) have reported increased resting concentrations of muscle creatine, creatine phosphate, and ATP after training, which were significantly reduced during subsequent 5- and 6-week periods of immobilization and training stoppage, respectively. It has also been suggested that changes in phosphate content and splitting mechanisms may be significant for sustaining the energy turnover need during high short-term power output activities (5). It is also likely that the decreases in maximal anaerobic power may be because of concomitant DTR of the acid-base buffering system, which may contribute to some extent to reduction in power output as the muscle becomes more acidotic.

## Practical Applications

Six weeks of detraining in recreationally trained men showed significant reductions in peak isometric torque production of the elbow extensor and flexor muscles, whereas maximal isometric and 1RM strength and vertical jump performance can be maintained during shorts periods of training stoppage. However, short-term (3 weeks) training cessation appears to significantly reduce the anaerobic power performance as measured by the Wingate test. All of these occurred in the absence of changes in resting serum hormonal concentrations. These data advocate the use of minimal RT maintenance programs over 6 weeks, which may well offset any detrimental changes in the recreational lifter.

## References


1. Colliander, E.B., and P.A. Tesch. Effects of detraining following short term resistance training on eccentric and concentric muscle strength. *Acta Physiol. Scand.* 144:23–29. 1992.
2. Cote, C., J.A. Simoneau, P. Lagasse, M. Boulay, M.C. Thibault, M. Marcotte, and C. Bouchard. Isokinetic strength training protocols: Do they induce skeletal muscle fiber hypertrophy? *Arch. Phys. Med. Rehabil.* 69:281–285. 1988.
3. Dudley, G.A., P.A. Tesch, B.J. Miller, and P. Buchanan. Importance of eccentric actions in performance adaptations to resistance training. *Aviat. Space Environ. Med.* 62:543–550. 1991.
4. Fleck, S.J., and W.J. Kraemer. *Designing Resistance Training Programs* (2nd ed.). Champaign, IL: Human Kinetics, 1997.
5. Green, H.J., J.A. Thomson, B.D. Daub, and D.A. Ranney. Biochemical and histochemical alterations in skeletal muscle in man during a period of reduced activity. *Can. J. Physiol. Pharmacol.* 58:1311–1316. 1980.
6. Häkkinen, K., M. Alen, and P.V. Komi. Changes in isometric force- and relaxation-time, electromyographic and muscle fibre characteristics of human skeletal muscle during strength training and detraining. *Acta Physiol. Scand.* 125:573–585. 1985.
7. Häkkinen, K., and P.V. Komi. Electromyographic changes during strength training and detraining. *Med. Sci. Sports Exerc.* 15: 455–460. 1983.
8. Häkkinen, K., and P.V. Komi. Changes in electrical and mechanical behavior of leg extensor muscles during heavy resistance strength training. *Scand. J. Sport Sci.* 7:55–64. 1985.
9. Häkkinen, K., P.V. Komi, and M. Alen. Effect of explosive type strength training on isometric force- and relaxation-time, electromyographic and muscle fibre characteristics of leg extensor muscles. *Acta Physiol. Scand.* 125:587–600. 1985.
10. Häkkinen, K., A. Pakarinen, M. Alen, H. Kauhanen, and P.V. Komi. Relationships between training volume, physical performance capacity, and serum hormone concentrations during prolonged training in elite weight lifters. *Int. J. Sports Med.* 8(Suppl.):61–65. 1987.
11. Häkkinen, K., A. Pakarinen, M. Alen, and P.V. Komi. Serum hormones during prolonged training of neuromuscular performance. *Eur. J. Appl. Physiol.* 53:287–293. 1985.
12. Häkkinen, K., A. Pakarinen, P.V. Komi, T. Ryushi, and H. Kauhanen. Neuromuscular adaptations and hormonal balance in strength athletes, physically active males, and females during intensive strength training. In: *Proceedings of the XII International Congress of Biomechanics No. 8.* R.J. Gregor, R.F. Zernicke, and W.C. Whiting, eds. Champaign IL: Human Kinetics, 1989. pp. 889–894.
13. Häkkinen, K., A. Pakarinen, H. Kyrolainen, S. Cheng, D.H. Kim, and P.V. Komi. Neuromuscular adaptations and serum hormones in females during prolonged power training. *Int. J. Sports Med.* 11:91–98. 1990.
14. Hather, B.M., P.A. Tesch, P. Buchanan, and G.A. Dudley. Influence of eccentric actions on skeletal muscle adaptations to resistance training. *Acta Physiol. Scand.* 143:177–185. 1991.
15. Hortobagyi, T., J.A. Houmard, J.R. Stevenson, D.D. Fraser, R.A. Johns, and R.G. Israel. The effects of detraining on power athletes. *Med. Sci. Sports Exerc.* 25:929–935. 1993.
16. Housh, T.J., D.J. Housh, J.P. Weir, and L.L. Weir. Effects of eccentric-only resistance training and detraining. *Int. J. Sports Med.* 17:145–148. 1996.
17. Houston, M.E., E.A. Froese, S.P. Valeriote, H.J. Green, and D.A. Ranney. Muscle performance, morphology and metabolic capacity during strength training and detraining: A one leg model. *Eur. J. Appl. Physiol.* 51:25–35. 1983.
18. Ishida, K., T. Moritani, and K. Itoh. Changes in voluntary and electrically induced contractions during strength training and detraining. *Eur. J. Appl. Physiol.* 60:244–248. 1990.
19. Jackson, A.S., M.L. Pollock, and A. Ward. Generalized equations for predicting body density in women. *Med. Sci. Sports Exerc.* 12:175–182. 1980.
20. Jaric, S., D. Ristanovic, and D.M. Corcos. The relationship between muscle kinetic parameters and kinematic variables in a complex movement. *Eur. J. Appl. Physiol.* 59:370–376. 1989.
21. Klausen, K., L.B. Andersen, and I. Pelle. Adaptive changes in work capacity, skeletal muscle capillarization and enzyme levels during training and detraining. *Acta Physiol. Scand.* 113: 9–116. 1981.

22. KRAEMER, W.J. Endocrine responses to resistance exercise. *Med. Sci. Sports Exerc.* 20(Suppl.):S152–S157. 1988.
23. KRAEMER, W.J., AND A.C. FRY. Strength testing: Development and evaluation of methodology. In: *Physiological Assessment of Human Fitness.* P.J. Maud and C. Foster, eds. Champaign, IL: Human Kinetics, 1995. pp. 115–138.
24. KRAEMER, W.J., L. MARCHITELLI, D. MCCURRY, R. MELLO, J.E. DZIADOS, E. HARMAN, P. FRYKMAN, S.E. GORDON, AND S.J. FLECK. Hormonal and growth factor responses to heavy resistance exercise protocols. *J. Appl. Physiol.* 69:1442–1450. 1990.
25. LINOSSIER, M.T., D. DORMOIS, C. PERIER, J. FREY, A. GEYSSANT, AND C. DENIS. Enzyme adaptations of human skeletal muscle during bicycle short-sprint training and detraining. *Acta Physiol. Scand.* 161:439–445. 1997.
26. MACDOUGALL, J.D., G.C.B. ELDER, D.G. SALE, J.R. MOROZ, AND J.R. SUTTON. Effects of strength training and immobilization on human muscle fibres. *Eur. J. Appl. Physiol.* 43:25–34. 1980.
27. MACDOUGALL, J.D., G.R. WARD, D.G. SALE, AND J.R. SUTTON. Biochemical adaptation of human skeletal muscle to heavy resistance training and immobilization. *J. Appl. Physiol.* 43:700–703. 1977.
28. MIKINES, K.J., B. SONNE, B. TRONIER, H. GALBO. Effects of acute exercise and detraining on insulin action in men. *J. Appl. Physiol.* 66:2080–2085. 1989.
29. NARICI, M.V., G.S. ROI, L. LANDONI, A.E. MINETTI, AND P. CERRETELLI. Changes in force, cross-sectional area and neural activation during strength training and detraining of the human quadriceps. *Eur. J. Appl. Physiol.* 59:310–319. 1989.
30. PAKARINEN, A., M. ALEN, K. HÄKKINEN, AND P.V. KOMI. Serum thyroid hormones, thyrotropin and thyroxine binding globulin during prolonged strength training. *Eur. J. Appl. Physiol.* 57:394–398. 1988.
31. POLLOCK, M.L., L. GARZARELLA, AND J.E. GRAVES. The measurement of body composition. In: *Physiological Assessment of Human Fitness.* P.J. Maud and C. Foster, eds. Champaign, IL: Human Kinetics, 1995. pp. 167–204.
32. SALE, D.G. Testing strength and power. In: *Physiological Testing of the High-Performance Athlete.* J.D. MacDougall, H.A. Wenger, and H.J. Green, eds. Champaign, IL: Human Kinetics, 1991. pp. 21–106.
33. SIMONEAU, J.A., G. LORTIE, M.R. BOULAY, M. MARCOTTE, M.C. THIBAULT, AND C. BOUCHARD. Effects of two high-intensity intermittent training programs interspaced by detraining on human skeletal muscle and performance. *Eur. J. Appl. Physiol.* 56: 516–521. 1987.
34. SIRI, W.E. Body composition from fluid spaces and density: Analysis of methods. In: *Techniques For Measuring Body Composition.* J. Brozek and A. Henschel, eds. Washington, DC: National Academy of Science, 1961. pp. 223–244.
35. STARON, R.S., F.C. HAGERMAN, AND R.S. HIKIDA. The effects of detraining on an elite power lifter: A case study. *J. Neurol. Sci.* 51:247–257. 1981.
36. STARON, R.S., M.J. LEONARDI, D.L. KARAPONDO, E.S. MALICKY, J.E. FALKEL, F.C. HAGERMAN, AND R.S. HIKIDA. Strength and skeletal muscle adaptations in heavy-resistance-trained women after detraining and retraining. *J. Appl. Physiol.* 70:631–640. 1991.
37. THORSTENSSON, A. Observations on strength training and detraining. *Acta Physiol. Scand.* 100:491–493. 1977.
38. WEIR, J.P., D.J. HOUSH, T.J. HOUSH, AND L.L. WEIR. The effect of unilateral concentric weight training and detraining on joint angle specificity, cross-training, and the bilateral deficit. *J. Orthop. Sports Phys. Ther.* 25:264–270. 1997.

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